**N: 111 Pharmacology Blue Print for Final Exam**

**Chapter: 1**

Role of the food and drug administration-------------------------------------------------1

* Pharmaceutical companies need approval from the FDA to market drugs
* **Role of FDA (1.7)**
  + Center for Drug Evaluation and Research (CDER)
    - Controls whether drugs (Rx & OTC) can be used for therapy
  + Center for Biologics Evaluation and Research (CBER)
    - Regulates the use of biologics
  + Center for Food Safety and Applied Nutrition (CFSAN)
    - Oversees administration of herbal products and dietary supplements
  + In other words…
    - They implement rigorous research standards to ensure the safety of all pharmaceuticals/biologics/supplements used by the American public.

**Chapter: 2**

Difference between brand name drugs and their generic equivalent--------------1

* Pg 13
* Generics are less expensive
* **Differences Between Brand-Name Drugs and Their Generic Equivalents** (2.3)
  + Bioavailability
    - The physiologic ability of a drug to reach its target cells and produce its effect
    - Can be effected by inert ingredients and tablet compression
    - Measured by how long a drug takes to exert its effects
    - Criticality is relative to what the drug’s use is
    - Negative Formulary Lists
      * Lists of trade-name drugs that cannot be dispensed as generics
      * Controversial – difference in bioavailability and bioequivalence may sometimes affect patient outcomes; however, consumer advocacy groups are concerned with higher cost of trade-name drugs.
  + Price
    - Exclusive rights to produce a drug 🡪 no competition 🡪 prices generally quite high

**Chapter: 4**

Excretion of Medication------------------------------------------------------------------------1

* Pg 41
* Absorption 🡪 Distribution 🡪 Metabolism 🡪 Excretion
* Acidity/Alkalinity factors
* **Excretion of Medications** (4.6)
  + Process by which drugs are removed from the body
  + The rate at which medications are excreted determines their concentration in the bloodstream/tissues
    - Concentration determines duration of action
  + Primary site of excretion is the kidney
    - Filters on average 180L of blood per day
    - Some drugs are small enough to be passively filtered at the glomerulus
    - Larger and/or protein-bound drugs have to be actively secreted into the distal tubule in order to be excreted
    - Urine pH also influences drug excretion
      * + In patients who have overdosed on a medication (needs to be excreted ASAP) we can give another drug that changes urine pH in order to help the patient detox more quickly.
    - Kidney filtration mechanisms are less active in:
      * + Infants
        + Elderly
        + Patients with Renal Dz

These patients retain drugs for an extended time

KIDNEY FUNCTION LAB VALUES AND DOSING REGIMES MUST BE FOLLOWED CLOSELY and OFTEN!!!

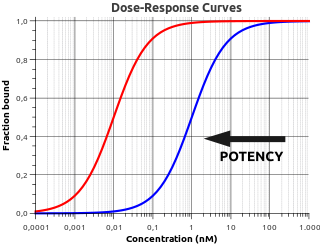
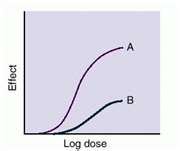
These patients are preferred to receive drugs that are eliminated through the feces

i.e. – biliary excretion

* + Drugs that change into gaseous forms can be excreted by the respiratory system
    - The better the patient’s gas exchange, the quicker the excretion.
  + Drugs can also undergo glandular excretion
    - Saliva, sweat, or breast milk

**Chapter: 5**

Potency and Efficacy-----------------------------------------------------------------------------1

* Pg 49
* Potency = strength per amount
* Efficacy = greater impact (this one is more desirable)
* **Potency and Efficacy** (5.4)
  + Within a pharmacological class not all drugs are equally effective; there are two ways to compare drugs within the same class
    - Potency
      * A drug that is more potent will produce a therapeutic effect at a lower dose
        + Therefore, in the graph, the Red Drug is more potent than the Blue Drug because the same effect is achieved with lower concentration.
    - Efficacy
      * The magnitude of maximal response that can be produced from a drug corresponds to its efficacy
      * i.e.: Morphine has higher efficacy than aspirin for severe pain to have the same impact
        + Therefore, in the graph, A would be morphine and B would be aspirin (effect is greater, even though dose is lower)
  + From a pharmacotherapeutic standpoint, efficacy is more important than potency – it’s the ability to relieve symptoms that prevails as the more important factor (that said, if two drugs are equivalent in efficacy, it is likely better to use the more potent one – which produces the same effect but with a smaller dose)

**Chapter: 6**

Steps of nursing process-----------------------------------------------------------------------1

* Know which comes first, and the sequence
* **The Nursing Process in Pharmacology**
  + The nursing process is essential to pharmacology
  + It is a systematic method of problem-solving
  + Forms the foundation of all nursing practice
  + Is essentially five steps:
    - **Assessment**
      * The systematic collection, organization, validation, and documentation of patient data
      * Begins with the nurse’s initial contact with the patient and continues with every contact
      * Gathering of baseline data
        + Subjective

“I have a 9 out of 10 headache.”

Take what the patient is saying seriously

* + - * + Objective

Physical assessment

Vital Signs

Height

Weight

Laboratory Values

Hepatic/Renal/Electrolyte Values all very important

* + - * Health History (tailored to the patient’s clinical condition) (Table 6.1)
        + Chief Complaint
        + Allergies
        + Past medical History
        + Family History
        + Medication history/OTC/herbal
        + Health management
        + Reproductive history
        + Personal-social history (alcohol/tobacco/caffeine)

Alcohol intake is important to know, as it may affect medications

* + - * + Health risk history
        + What is *not* being said may be as important as what *is* being said
        + The effectiveness of drug therapy must be evaluated

If drugs aren’t providing desired therapeutic effects, more assessment is necessary to determine why

Any adverse effects must also be assessed (including follow-up vitals and labs)

* + - * An assessment of the ability of the patient to assume responsibility for self-administration of medications is necessary
        + Make sure the patient knows how to take his medications – have him repeat it back to you, preferably at multiple occasions
      * Factors which have to be evaluated are the patient’s:
        + Financial ability to afford Rx
        + Physical ability to dispense (open bottles, etc.)
        + Ability to understand dosing and intended effect
        + Ability to contend with:

Intended effects

Side effects

* + - **Diagnosis**
      * Once the baseline data has been gathered, a Nursing Diagnosis is made
      * NANDA definition:
        + A clinical judgment about individual, family, or community responses to actual or potential health/life processes. Per NANDA, Nursing Diagnoses provide the basis for selection of nursing interventions to achieve outcomes for which the nurse is accountable
        + In other words:

Nursing diagnosis focuses on a patient’s response to a health or life process, and are used as the basis for establishing goals and outcomes

* + - * Goals: what the patient will be able to achieve
      * Outcomes: objective measurement of these goals
        + Goals and Outcomes must be prioritized based on assessment data and nursing diagnoses: i.e. – relief of pain is a priority over nausea
        + Example:

While assessing a patient, he asks you questions which indicate his lack of understanding about the importance of his medication

Nursing Diagnosis: **Knowledge Deficit** related to drug therapy (Table 6.2 has more diagnoses)

Goal: for the patient to demonstrate an understanding of the drug’s action

Outcome: the patient accurately describing the drug’s action and side-effects

* + - * In terms of pharmacotherapy, the diagnosis phase of the nursing process addresses these main areas:
        + Promoting therapeutic drug effects
        + Minimizing adverse drug effects and toxicity
        + Maximizing the ability of the patient for self-care, including the knowledge, skills, and resources necessary for safe and effective drug administration
    - **Planning**
      * Care Plans
        + Once the Nursing Diagnosis has been made and the goals and outcomes determined, a Nursing Care Plan is formulated
        + The Care Plan describes the steps that will be followed to reach the desired goals and outcomes
      * In terms of pharmacotherapy, the planning phase of the nursing process involves 2 main components:
        + Drug administration
        + Patient Teaching
      * Hint: Use Nursing Process Focus and Nursing Dx in the textbook (end of each chapter)
        + They will guide us to what is important and unique about the drugs used, and will help formulate patient teaching
    - **Implementation**
      * Putting the plan into *action*
        + i.e.: administering a drug; teaching the patient about the drug and its side-effects, etc.
      * This phase also includes monitoring the effects of the teaching, and of the drug itself
      * Both effectiveness, and adverse effects
      * A thorough knowledge of the actions of each medication is necessary to carry out this monitoring process.
      * Teaching is a primary role for nurses
        + JCAHO gives it weight in law… it is of key importance in accreditation standards
        + Every nurse-patient interaction can present an opportunity for teaching, which should be taken advantage of because smaller portions of education over time are more effective than cramming it all into one session.
        + Areas of Patient Teaching on medications:

Therapeutic use and outcomes

Monitoring side effects and adverse effects

Medication administration

Other monitoring and special requirements

* + - * Note on Pediatrics
        + Be sure to note differences in dosing from adult standards – small errors can have serious consequences
    - **Evaluation**
      * Evaluates the effectiveness of the implementation of the Care Plan
      * Compares current status with desired outcome
      * The process comes full circle, as the nurse reassesses the patient, and changes the goals and outcomes accordingly
      * Evaluation is not the end of the cycle, but the beginning of the next cycle
      * Partially met goals require continued interventions
        + i.e. – increasing the drug dose to achieve therapeutic levels

**Chapter: 7**

Pharmacotherapy of preschoolers: Best method of medication administration--------1

* **Pharmacotherapy of Preschoolers and School-Age Children (7.7)**
  + Age 3-5 years old – the child begins to refine motor skills and develop language abilities,
  + Give short, concrete explanations to toddlers followed immediately by administration
    - Provide physical comfort (touch, hug, verbal praise) following administration
  + Mix bad-tasting meds with small amount of jam, syrup, or fruit puree
    - Don’t use milk, OJ, or cereal or child might associate healthy things with bad taste
  + IM meds 🡪 can be given in the ventrogluteal muscle after the child has walked for a year; otherwise the vastus lateralus muscle is still used
  + IV meds 🡪 peripheral veins
  + Restraint may be necessary at this age
  + Sit them on your lap and give a brief explanation.
  + Afterwards, the child may benefit from acting out the situation with a doll, where the child plays the role of the doctor or nurse and gives the “sick” doll a pill or injection
    - This can help them feel safer and more in control

**Chapter: 8**

Gender influences on pharmacotherapy---------------------------------------------------1

* **Gender influence on pharmacotherapy**
  + Women tend to pay more attention to changes in their health and seek medical attention sooner
  + Up to 3x as many women suffer from Alzheimer’s
  + Adherence to medication regimens can be influenced by gender-specific side-effects
    - some antihypertensives can cause/worsen male impotence
    - several drugs can cause gynecomastia (embarrassing to men)
    - some medications cause masculinizing effects (embarrassing to women)
    - BCP causes increased risk of thromboembolytic disorders
  + Physiological differences between men and women:
    - Fat-to-muscle ratio
    - Cerebral blood flow (response to analgesics)
    - Elimination rates (slower elimination of benzodiazepines in women, exacerbated by BCP)
  + Past drug research studies were only conducted on men
  + Gender inequity is a resolving issue

**Chapter: 9**

Figure 9.1 Categorizing medication errors-------------------------------------------------------1

* Know Category A, B, and G
  + - **Category A** – Circumstances or events that had potential to cause an error
    - **Category B** – Error that did not reach the patient (an “error of omission” does reach the patient)
    - Category C – Error that reached the patient but did not cause harm
    - Category D – Error that reached the patient and required monitoring to confirm that it resulted in no harm to the patient and/or required intervention to preclude harm
    - Category E – Error that may have contributed to or resulted in temporary harm to the patient and required intervention
    - Category F – Error that may have contributed to or resulted in temporary harm to the patient and required initial or prolonged hospitalization
    - **Category G** – Error that may have contributed to or resulted in permanent patient harm
    - Category H – Error occurred that required intervention necessary to sustain life
    - Category I – Error that may have contributed to or caused patient death

**Chapter: 13**

* Know the diagram on Pg 128 before you can begin to answer any of these questions!!!

|  |  |  |
| --- | --- | --- |
|  | **SNS** | **PSNS** |
| **Pupils** | Dilates | Constricts |
| **Salivary Glands** | Inhibits | Stimulates |
| **Heart** | Accelerates | Slows |
| **Bronchioles** | Dilates | Constricts |
| **Digestive System** | Inhibits | Stimulates |
| **Gallbladder/Liver** | Hepatic glycogenolysis | Gall bladder stimulation |
| **Adrenals** | Secretes NE and Epi | - |
| **Bladder** | Relaxes | Contracts |
| **Sex Organs** | Inhibits (but promotes ejaculation) | Stimulates |

Autonomic nervous system--------------------------------------------------------------------1

* **Autonomic NS**
  + A division of the peripheral NS
  + Involuntary control over smooth/cardiac muscle and glands

Sympathetic nervous system------------------------------------------------------------------1

* **Sympathetic Nervous System (SNS)**
  + Fight or flight
  + Activation under stressful conditions
  + Dilates pupils, inhibits salivation, accelerates heart, dilates bronchioles, inhibits digestion, stimulates release of glucose, secretes epinephrine/norepinephrine, relaxes bladder, inhibits sex organs

Adrenergic receptor activation/agonist---------------------------------------------------- 1

* (Table 13.2) Pg 132
  + **Albuterol**
    - Beta2 receptor subtype
      * Effect: inhibition of smooth muscle
      * Location: all sympathetic organs except the heart (bronchioles, arterioles, visceral organs)
      * (beta**2**=two lungs)
      * (Also used for decongestion, COPD, Slowing of uterine contractions)
    - Primarily used in asthma
      * Bronchodilator (Beta2 specific sympathomimetic)
  + Other receptor types
    - alpha1
      * Effects: vasoconstriction, dilation of pupils
      * Locations: all sympathetic organs except the heart
      * Uses:
        + Treats hypertension (effect in the brainstem), Treats shock, nasal congestion
    - alpha2
      * Effect: inhibition of release of NE
      * Location: presynaptic adrenergic nerve terminals
      * Uses:
        + Treats hypertension (effect in the brainstem), Causes sedation (CNS), Treats nasal congestion
    - beta1
      * Effects: increased heart rate and force of contraction; release of renin
      * Locations: Heart and kidneys
      * (beta**1** =one heart)
      * Uses:
        + Cardiac arrest, heart failure, or shock

Adrenergic antagonist --------------------------------------------------------------------------1

* (Table 13.3)
  + **Carvedilol**
    - Alpha1, beta1 and beta2 receptor subtypes (in other words, it is NOT cardioselective)
    - Primarily used for hypertension and angina
  + **Atenolol** 
    - Beta1 receptor subtypes (in other words, it is cardioselective)
    - Primarily used for hypertension and heart failure

Beta Agonist/use--------------------------------------------------------------------------------- 1

* **Albuterol**
  + Used for asthma
* Other uses: decongestion, slowing uterine contractions, treating COPD, treating cardiac arrest, treating heart failure, cardiac arrest, and shock

Beta blockers/precautions----------------------------------------------------------------------1

* **Precautions**
  + Because they are used to lower blood pressure, they can lower it too much!
  + Change positions slowly, avoid caffeine, avoid alcohol and hazardous activities, report side effects, don’t stop abruptly
  + Assess VS (BP and HR) before administering (don’t give if BP or HR are low!!!)
  + Assess history of COPD/asthma, hypotension, dysrhythmias, HF
    - If it is not cardioselective, it may cause bronchoconstriction

Beta blockers atenolol, metoprolol (selective)/adverse effects -----------------------1

* **Beta blockers atenolol, metoprolol (selective)/adverse effects:**
  + Selective = heart-specific side effect
  + Side Effects: Bradycardia, heart failure, pulmonary edema, hypotension, fatigue, dizziness, depression, lethargy, NVD (nausea/vomiting/diarrhea), impotence
* Suddenly stopping beta blockers 🡪 hypertension, tachycardia

Anti-cholinergic drug/contraindication ------------------------------------------------------1

* **Clinical Applications of Anticholinergics** (13.10)
  + Anticholinergics – inhibit PSNS impulses
  + This suppression induces a fight-or-flight response
    - Dilation of pupils, urinary retention, slowing of GI motility, increase in heart rate, drying of secretions, relaxation of bronchi
  + Historical example of an anticholinergic = belladonna “pretty woman” – women used to apply it to their faces to get rosy cheeks and dilated, doe-like eyes…
  + The most accurate term for this class is muscarinic antagonists because they are selective for ACh muscarinic receptors (and have little effect on nicotinic receptors)
  + They act directly by competing with ACh for binding muscarinic receptors
  + Therapeutic uses (try and figure out why based on known effects):
    - GI disorders, Ophthalmic procedures, Cardiac rhythm abnormalities, preanesthesia, asthma
  + Examples:
    - Atropine (prototype drug)
      * Antidote for cholinergic agent poisoning (medications/pesticides/poisonous mushrooms)
    - Deltrol
      * Helps with urinary incontinence (“Gotta-go-gotta-go” commercial)
    - Scopolamine (Hyoscine)
      * Produces sedation; prevents motion sickness
    - Benztropine (Cogentin)
      * Reduces muscular tremors/rigidity of Parkinson’s disease
    - Ipratropium (Atrovent)
      * Safer than many other anticholinergics, because it is applied as an aerosol spray (producing more local effects)
      * Used to treat COPD
  + Side Effects:
    - High incidence
    - Small doses decrease HR but larger doses INCREASE HR
    - Tachycardia, dysrhythmias, ischemia (restriction of blood supply), CNS stimulation, constipation, urinary retention in men with prostate disorders, dry mouth, dry eyes, sweating inhibition (heat stroke!), photophobia (due to pupil dilation), decreased bronchial secretions
  + Overdose (anticholinergic crisis) symptoms:
    - Fever, visual changes, difficulty swallowing, psychomotor agitation, hallucinations
    - “Hot as hades, blind as a bat, dry as a bone, mad as a hatter.”
  + Nursing Implications:
    - Assess for allergies, BPH, glaucoma, tachycardia, MI, CHF, hiatal hermia, GI or GU obstruction
    - Take baseline VS
    - Overdose can be life-threatening
    - Blurred vision side effect can be dangerous if driving/operating machinery
    - Apply sunglasses/sunscreen for photosensitivity
    - Apply pressure to inner canthus (the medial corner of the eye over the nasolacrimal duct) to prevent systemic absorption
    - Monitor side-effects
  + Contraindications:
    - Glaucoma, acute hemorrhage, tachycardia, GI obstruction

Cholinergic agonist/side effect/myasthenia gravis----------------------------------------1

* Parasympathomimetics – stimulate the PSNS and produce symptoms of rest-and-digest response
  + Increase secretions (including salivation & sweating) and peristalsis, increase urinary frequency, constrict pupils, reduce intraocular pressure
* Acetylcholine is an NT in the SNS, PSNS, and skeletal muscle… so obviously drugs like ACh (cholinergic agents) will have widespread/varied effects
* Subclasses
  + Direct-acting agents
    - i.e. – bethanechol (Urecholine)
    - Bind to cholinergic receptors to produce rest-and-digest response
    - They have longer-lasting effects (because they are more resistant to acetylcholinesterase (the enzyme that breaks down ACh))
    - Direct-acting agents = moderately selective = **muscarinic agonists**
  + Indirect-acting agents
    - i.e. – neostigmine (Prostigmin)
    - They inhibit the action of acetylcholinesterase (AChE) allowing endogenous (natural) ACh to not be destroyed!
    - In other words, they help prolong the action of the body’s own ACh
    - Indirect-acting agents = nonselective = **cholinesterase inhibitors**
    - Used to treat myasthenia gravis (an AI disease which leads to destruction of nicotinic receptors in skeletal muscles)
      * Because direct-acting cholinergic agents are more selective for muscarinic receptors, we can bet that drugs for myasthenia gravis (a nicotinic receptor related disorder) will be indirect-acting cholinergic agents!
      * Administration of cholinergic agents (pyridostigmine or neostigmine) stimulates skeletal muscle contraction, which helps reverse symptoms of severe muscle weakness
      * It can be administered before meals to help patients chew their food
    - Side Effects:
      * “SLUDGE”
      * Salivation, Lacrimation, Urinary incontinence, Diarrhea, Gastrointestinal cramps, Emesis
  + #1 cholinergic inhibitor – narcotics (they cause diarrhea)

**Chapter: 18**

Nonpharmacological Techniques for Pain Management ----------------------------------1

* Provide the opportunity to give lower doses of pharmaceuticals (leading to fewer side effects)
  + Techniques:
    - Acupuncture
    - Biofeedback Therapy
    - Massage
    - Heat or cold packs
    - Meditation or prayer
    - Relaxation therapy
    - Art or music therapy
    - Imagery
    - Chiropractic manipulation
    - Hypnosis
    - Therapeutic or physical touch
    - Transcutaneous electrical nerve stimulation (TENS)
    - Energy therapies (i.e. - Reiki and Qi Gong)

Neural Mechanism of control/Substance P / Aδ and C fiber------------------------------1

* Pain transmission begins when pain receptors (nocicpetors – free nerve endings) are stimulated
* The pain impulse is sent to the spinal cord by Aδ and C fibers.
  + Aδ fibers – lightly myelinated (fast)
    - Signal sharp, well-defined pain
  + C fibers – unmyelinated (slow)
    - Signal dull, poorly-localized pain
* From the spinal cord the message is transmitted to the brain by a NT called **substance P**
  + Spinal NT (substance P) is critical – it **controls whether or not pain is detected by the brain**

Treatment for opioid dependence---------------------------------------------------------------1

* + People who abuse opioids quickly become tolerant to euphoric effects and subsequently increase dose/frequency. When physically dependent patients attempt to discontinue drug use, they experience extremely uncomfortable symptoms, so this convinces many people to continue using, so as to avoid this suffering.
  + Physical dependence can be overcome by stopping for 7 days, but psychological dependence can occur up to years following discontinuation. This means that significant support groups are paramount.
  + **Treatment:**
    - Switching dependent patients to methadone (Dolophine), which does not cause euphoria.
      * The patient must then continue taking methadone (to avoid withdrawal symptoms) until the patient decides to enter a total withdrawal treatment program
      * This method allows patients to return to functioning without the physical, emotional, and criminal risks of illegal drug use
    - Administering buprenorphine (Subutex) sublingually
      * This is a mixed opioid agonist-antagonist
      * It prevents opioid withdrawal symptoms
  + The patient is later switched to a buprenorphine-naloxone combination for maintenance

Narcotic: Morphine/mechanism of action/side effects-------------------------------------1

* **Prototype: Morphine**
  + **Mechanism of action**
    - Binds with mu and kappa receptors
  + Effects:
    - Euphoria
    - Constriction of the pupils
    - Stimulation of cardiac muscle
  + Use
    - Relief of serious acute/chronic pain
    - Preanesthetic medication
    - Relieve shortness of breath associated with
      * MI
      * HF
      * Pulmonary edema
  + **Adverse Effects**
    - Dysphoria (restlessness, depression, anxiety)
    - Hallucinations
    - Nausea
    - Constipation
    - Dizziness
    - Itching
    - Cross tolerance to other opioids
    - **Underlined effects in Table 18.2**
      * Anaphylactoid reaction, cardiac arrest, severe respiratory depression or arrest, convulsions
  + Overdose
    - Severe respiratory depression
    - Cardiac arrest
  + Contraindications
    - Gallbladder disease
      * Intensify or mask the pain
    - Acute/severe asthma
    - GI obstruction
    - Severe hepatic or renal impairment

Opioid Antagonist action/mechanism-----------------------------------------------------------1

* **Opioid Overdose treatment**
  + IV naloxone (Narcan) (most preferred) – opioid antagonist
    - Mechanism of action: blocks mu and kappa receptors
  + Other treatments:
    - Activated charcoal
    - Laxatives

Pharmacotherapy with NSAIDS-------------------------------------------------------------------1

* Table 18.3
* Underlined effects of Ibuprofen/naproxen/etc
  + Aplastic anemia, drug-induced peptic ulcer, GI bleeding, agranulocytosis, laryngospasm, laryngeal edema, peripheral edema, anaphylaxis, acute renal failure, vomiting, constipation, diarrhea
* Monitor CBC and renal function tests (Cr, BUN)

Classification of Opioid receptor------------------------------------------------------------------1

* Opioid receptor types:
  + Mu (types 1 and 2)
    - Analgesia, decreased GI motility, euphoria, respiratory depression, sedation, physical dependence
  + Kappa
    - Analgesia, decreased GI motility, sedation, miosis
  + Sigma
  + Delta
  + Epsilon

Opioid adverse effects ------------------------------------------------------------------------------1

* Most important/dangerous is respiratory depression
* Constipation
* Sedation
* Nausea
* Orthostatic hypotension

Migraine Headache/Sumatriptan/Adverse effect--------------------------------------------- 1

* Used for vasoconstriction to relieve headache – in the Triptan class
* Adverse effects in Table 18.4
  + Coronary artery vasospasm, MI, cardiac arrest
  + Asthenia, tingling, warming sensation, dizziness, vertigo
* Don’t give to patients with angina

**Chapter 33**

Role of chemical mediators in inflammation----------------------------------------------------1

* Chemical mediators are “alarms” that notify surrounding area of an injury
* Examples:
  + **Histamine**
    - * Stored/released by mast cells
      * Causes vasodilation, smooth muscle constriction, swelling, and itching
    - Leukotrines
      * Stored/released by mast cells
      * Effects similar to histamine
    - Bradykinin
      * Present in inactive form in plasma and mast cells
      * Causes vasodilation 🡪 pain
      * Effects similar to histamine
    - Complement
      * Series of proteins that combine and cascade to neutralize/destroy antigens
    - Prostaglandins
      * Stored/released by mast cells
      * Present in most tissues
      * Increases capillary permeability, attracts WBC to site, and causes pain
  + Mast cells = cells in connective tissue that detect inflammation and release histamine to initiate the inflammatory response
    - This causes plasma, complement proteins, and phagocytes to flood the area and neutralize foreign agents
  + Drugs that are histamine receptor antagonists are used to treat allergic rhinitis (stop inflammation from occurring
* Rapid release of chemical mediators 🡪 ANAPHYLAXIS

NSAIDs/labs to monitor/maximum dose/adverse effects/Interactions-------------------3

* Table 33.2
  + Common side effect = nausea/heartburn/stomach pain(dyspepsia)/diarrhea
    - Aspirin – tinnitus, prolonged bleeding time
    - Celebrex – headache, Pharyngitis, rash
    - Ibuprofen etc. – vomiting, dizziness
* **Treating Inflammation with NSAIDs** (33.4)
  + NSAIDs have analgesic, antipyretic, and anti-inflammatory properties, and are prescribed for mild to moderate inflammation
  + They have a relatively high safety margin and are available OTC
  + Different NSAIDs have about the same efficacy, but vary in their adverse effects
  + Note: acetaminophen is NOT an NSAID – it has no anti-inflammatory action (even though it is an analgesic/antipyretic)
  + NSAIDs can differ in their duration of action, which is important for longterm users
  + They can also produce varied responses – some patients respond better than others to a particular drug
  + Action of NSAIDs:
    - They inhibit the synthesis of prostaglandins
      * Prostaglandins = Lipids that promote inflammation, among other potent physiologic effects
    - They do so by inhibiting the enzyme that helps make prostaglandins – cycolooxygenase (COX)
  + COX subtypes:
    - COX-1 – present in all body tissues and serves protective functions:
      * Reducing gastric acid secretions
      * Promoting renal blood flow
      * Regulating smooth muscle tone in blood vessels and the bronchial tree
    - COX-2 – formed only after tissue injury
      * Promotes inflammation
    - First generation NSAIDs (ibuprofen, aspirin) blocked both COX subtypes
      * However, inhibiting COX-1 produces undesirable side effects (bleeding, gastric upset, reduced kidney function)…
  + **Salicylates**
    - Aspirin – a long-used nonspecific COX inhibitor
      * (Prevents COX enzymes from forming inflammatory prostaglandins)
    - It has a cardioprotective effect – preventing clot formation and strokes
      * In fact, a single dose of aspirin can inhibit a platelet for its entire 8-11 day lifespan…
        + Therefore, we must monitor patients for bleeding!
    - It is only recommended for mild inflammation, because higher doses may result in a high incidence of side-effects:
      * Epigastric pain, heartburn, stomach bleeding related to ulceration
      * Enteric coating minimizes these effects
    - High doses cause salicylism:
      * Tinnitus, dizziness, headache, excessive sweating
  + **Ibuprofen and Ibuprofen-like NSAIDs**
    - Developed as aspirin alternatives as nonspecific COX inhibitors
    - These NSAIDs can affect platelet function, but they have a lower risk than with aspirin
  + **Selective COX-2 Inhibitors**
    - Because they do not inhibit COX-1, they do not produce adverse effects on the GI system, and do not affect coagulation ☺
    - However, after postmarketing data revealed that rofecoxib (Vioxx) doubled the risk of heart attack and stroke in those taking it for extended periods, the number of COX-2 inhibitors on the market was reduced to only 1 – celecoxib (Celebrex)
    - Celecoxib is used as an anti-inflammatory
      * It is also used to reduce the number of colorectal polyps in adults with FAP (familial adenomatous polyposis)
* **Labs to Monitor**
  + BUN/creatinine (to assess kidney function)
  + Liver enzymes (to assess for hepatic impairment)
  + CBC/Hb/hematocrit (to assess for blood loss)
  + Clotting profile (pt, ptt) (to assess for clotting impairments)
  + Serum salicylate levels (for aspirin, to assess for salicylism)
* **NSAIDs maximum doses**
  + Aspirin = 4g (4,000mg)/day
  + Ibuprofen = 3.2g (3,200mg)/day
  + Naproxen = 1g (1,000mg)/day

Treating acute or severe inflammation with glucocorticoid/adverse effect -------------2

* Adverse effects
  + Table 33.4
    - *Mood swings, weight gain, acne, facial flushing, nausea, insomnia, sodium and fluid retention, impaired wound healing, menstrual abnormalities*
    - Peptic ulcer, hypocalcemia, osteoporosis with possible bone gractures, loss of muscle mass, decreased growth in children, possible masking of infections (glucocorticoids cause immunosuppression)
  + Prototype Prednisone
    - Cushing’s syndrome (**hyperglycemia**, fat redistribution, muscle weakness, **bruising**, bones that easily fracture), gastric ulcers
  + Other: **Hypertension**
* Taper the dose
* Give only for limited amount of time

Fever/Acetaminophen/mechanism of action/Adverse effect/Interactions---------------3

* **Acetaminophen**
  + Therapeutic: antipyretics, nonopioid analgesics
  + Pharmacologic: centrally acting COX inhibitor (but it doesn’t have anti-inflammatory effects)
  + **Mechanism of Action**: it reduces fever at the level of the hypothalamus (by inhibiting prostaglandins which may serve as mediators of pain and fever) and dilation of peripheral blood vessels, which enables sweating and dissipation of heat.
  + **Adverse effects**: hepatotoxicity
  + **Interactions**
    - Interacts with warfarin, alcohol, other hepatotoxic drugs

Nursing Process: Anti-Inflammatory and Anti-Pyretic Therapy-Implementation----------1

* Minimizing adverse effects (Pg. 474)
  + Monitor labwork (hepatic and renal function tests, CBC, lytes, glucose, lipids, coagulation/bleeding time studies)
    - Aspirin and salicylates affect platelet aggregation
    - Acetaminophen can be hepatotoxic
    - Corticosteroids affect CBC, glucose, and lytes
  + Monitor for abdominal pain, black or tarry stools, blood in the stool, hematemesis or coffe-ground emesis, dizziness, and hypotension (esp w/ tachycardia)
    - NSAIDs and glucocorticoids may cause GI bleeding
  + Monitor tinnitus, difficulty hearing, light-headedness, difficulty with balance
    - NSAIDs and salicylates may be ototoxic
  + Monitor urine output and renal function studies periodically
    - NSAIDs and salicylates may be renal toxic during long-term or high dose therapy

**CHAPTER 15 – Drugs for Seizures**

Benzodizepine: Table 15.3 Adverse effects-------------------------------------------------------1

* **Lorazepam, Diazepam**
  + Laryngospasm, respiratory depression, cardiovascular collapse, coma

Table 15.3 Barbiturates Adverse effects-----------------------------------------------------------1

* **Phenobarbital**
  + Agranulocytosis, SJS, angioedema, laryngospasm, respiratory depression, CNS depression, coma, death

Table 15.3 Newer GABA-Related Drugs Adverse effects--------------------------------------1

* **Gabapentin**
  + Serious disfiguring and debilitating rashes, sudden unexplained death in epilepsy (SUDEP), withdrawal seizures on discontinuation of drug

Prototype Phenobarbital; Adverse effect /Mechanism of action 1

* **Adverse Effect**
  + Vitamin deficiencies - Dvit, Folate (B9), B12
  + Laryngospasm
  + Drowsiness
* **Mechanism of Action**
  + Enhances the action of GABA (which is responsible for suppressing abnormal neuronal discharges that can cause epilepsy)

Table 15.4 Phenytoin like drug: adverse effect 1

* **Carbamazepine, Valproic Acid**
  + Agranulocytosis, aplastic anemias, bullous exfoliative dermatitis, SJS, toxic epidermal necrolysis, bone marrow depression, acute liver failure, pancreatitis, heart block, respiratory depression

Prototype drug Phenytoin: administration alerts, Interactions 1

* **Phenytoin**
  + Desensitizes sodium channels
  + Effective against most seizures except absence seizures
  + Most observed adverse effect: gingival hyperplasia
* **Administration Alerts**
  + When administering IV, mix with saline *only* and infuse slowly. Mixing with other meds or dextrose produces precipitate
  + Always flush IV lines with saline before hanging phenytoin as a piggyback (traces of dextrose can cause microscopic precipitate formation which become emboli if infused).
  + Use an IV line with a filter when infusing phenytoin
  + Never give IM – it will irritate the tissue
  + Give in large veins or via a central venous catheter
  + Avoid hand veins (may cause serious local vasoconstrictive response)
  + Pregnancy Category D
* **Interactions**
  + Drug-Drug
    - Oral anticoagulants
    - Glucocorticoids
    - H2 antagonists (such as Zantac or Pepcid)
    - Antituberculin drugs
    - Folic acid, calcium, and vitamin D
    - Impairs function of digitoxin, doxycyclin, furosemide, estrogens, oral contraceptives, theophylline
    - Tricyclic antidepressants – can trigger seizures!
  + Lab Tests
    - May increase glucose levels
  + Herbal
    - Herbal laxatives may increase potassium loss
    - Ginkgo may reduce its therapeutic effects

Prototype drug Ethosuximide action and uses 1

* **Actions and Uses**
  + Succinimides are generally only effective against absence (petit mal) seizures
  + Mechanism of action: delays the entry of calcium into neurons by blocking low-threshold calcium channels - thereby elevating the neuronal threshold
  + May be used in combination with other antiseizure meds to treat tonic-clonic or psychomotor seizures, but usually ineffective against these when used on its own

Patient receiving antiseizure drug therapy: Nursing Process-Implementation 1

* Ensuring Therapeutic Effects
  + Assess for therapeutic effects
  + *Frequency and severity of seizures should be diminished, although symptoms may not completely resolve*
* Minimizing Adverse Effects
  + Monitor vitals, mental status, coordination, and balance periodically
  + Monitor for fall risks
    - Monitor ambulation until drug effects are known
    - be particularly cautious with older adults at risk for falls
    - *Antiseizure med side-effects of drowsiness, dizziness, hypotension, and impaired mental/physical abilities may increase fall risk*
  + Take special precautions in pediatric patients
    - Monitor for developmental delays in children
      * Assess height, weight, and developmental level
      * Assess school performance
      * *Adverse effects of antiseizure meds may hinder normal growth & development*
    - Monitor for paradoxical response to barbiturates
      * *Hyperactivity may occur*
    - Assess for restlessness and agitation
      * *Valproic acid can cause an idiosyncratic response in children*
  + **Monitor blood levels**
    - Drug levels, CBC, renal & hepatic function, pancreatic enzymes
    - Instruct clients to wear/carry ID indicating presence of seizure disorder
    - ***Narrow therapeutic index on some drugs***
    - *Antiseizure meds may cause hepatotoxicity*
    - *Valproic acid may cause pancreatitis*
  + Monitor neurological function
    - Assess LOC, disorientation, confusion, agitation
    - *Neurologic symptoms may indicate overmedication or adverse effects*
  + Monitor visual symptoms
    - Assess visual acuity, blurred vision, loss of peripheral vision, seeing haloes around lights, acute eye pain
    - Also assess for N&V accompanying visual changes
    - *Benzodiazepines may cause increase in IOP in narrow-angle glaucoma patients*
  + **Monitor for bleeding/infection**
    - ***Antiseizure meds may cause blood dyscrasias and increased chances of bleeding/infection***
  + Monitor emotional status
    - *Antiseizure meds may increase the risk of depression and suicide*
    - Assess use of other CNS depressants
      * *Alcohol use or use of other CNS depressants may increase adverse effects*
  + **Monitor mouth**
    - Assess gums and oral hygeine
    - *Hydantoins and phenytoin-like drugs may cause* ***gingival hyperplasia,*** *increasing the risk of oral infections*
  + Monitor diet/supplements
    - Assess vitamin intake, caffeine and nicotine usage
    - *Caffeine and nicotine may decrease the effectiveness of benzodiazepines*
    - *Most antiseizure drugs affect the absorption of vitamins K, D, B’s, and folic acid*
  + **Assess possibility of pregnancy**
    - Risk, plans for it, breast-feeding, contraceptive use
    - ***Antiseizure meds are Category D***
    - *Barbiturates decrease effectiveness of oral contraceptives*
  + **Avoid abrupt discontinuation of therapy**
    - ***Status epilepticus may occur***
  + Assess home storage and ID risks for corrective action
    - Instruct clients that drugs should not be kept at bedside
    - *OD may occur if clients take additional doses when experiencing drowsiness/disorientation from medication effects.*
    - *OD may be fatal*
  + Provide emotional support and appropriate referrals PRN
    - *Social isolation and low self-esteem may occur with continued seizure disorder*
  + Monitor IV site
    - Assess for blanching, pain, irritation
    - *Benzodiazepines, hydantoins and barbiturates are irritating (especially note the administration alert of phenytoin)*
    - *Blanching/pain are indications of extravasation 🡪 d/c IV immediately!*

**Chapter 20 – Drugs for Degenerative Diseases of the Nervous system**

Table 20.2 Levadopa, Sinemet, ropinirole, Selegiline: Adverse Effects 2

* Acute MI, shock, neuroleptic malignant syndrome, agranulocytosis, depression with suicidal tendencies, EPS, fulminant liver failure, severe hepatocellular injury, *constipation, orthostatic hypotension, choreiform (rapid, jerky movements) and involuntary movements*

Prototype Drug Levadopa: Drug-Drug Interaction/action/administration alert 3

* **Action and uses**
  + Restores dopamine in extrapyramidal areas of the brain, thus relieving some Parkinson’s symptoms.
  + To increase its effect, it is often combined with carbidopa (which prevents its enzymatic breakdown)
  + Up to 6 months may be needed to achieve maximum therapeutic effects
* **Administration Alerts**
  + The patient may be unable to self-administer medication and may need assistance
  + Administer exactly as ordered
  + **Abrupt withdrawal of the drug can result in parkinsonism crisis or neuroleptic malignant syndrome (NMS)**
  + Pregnancy Category C
* Early signs of toxicity: blepharospasm (spasmodic eye winking) and muscle twitching
* **Drug-Drug Interaction**
  + Many!
  + Tricyclic antidepressants decrease its effects, increase postural hypotension, and may increase sympathetic activity (hypertension, sinus tach)
  + **MAOI** within 14-28 days? Do not take levodopa, or it may precipitate hypertensive crisis
  + Haloperidol may antagonize the therapeutic effects
  + Methyldopa may increase toxicity
  + Antihypertensives may cause increased hypotensive effects
  + Anticonvulsants may decrease the therapeutic effects
  + **Antacids (containing magnesium, calcium, or sodium bicarbonate)** may increase absorption, leading to toxicity
  + Pyridoxine reverses antiparkinsonism effects of levodopa

Table 20.3 Benztropine (Cogentin), Benadryl: Adverse effect 2

* Anticholinergic drugs and drugs with anticholinergic activity
  + Anticholinergic effects: think opposite of SLUDGE
* Adverse Effects:
  + Paralytic ileus, cardiovascular collapse, *tachycardia, hypotension*
    - Additional effects: *Sedation, nausea, constipation, dry mouth, blurred vision, drowsiness, dizziness, nervousness*
  + Note: tachycardia is anything over 100bpm
  + Patients on this medication have a decreased ability to tolerate heat
* Antidote: physostigmine salicylate

Nursing Process: Levadopa with carbidopa (implementation-Patient & family education) 1

* Teach that improvement may be gradual, but the patient should report increasing symptoms which may affect dosing
  + Encourage the patient to keep a symptom diary if the effects seem to be diminishing
  + *Increasing symptoms may indicate that the dose may need to be increased due to developed tolerance*
  + *Symptoms of blepharospasm and muscle twitching are early signs of levodopa toxicity*
* Instruct patient to call for assistance when getting out of bed or walking alone if Parkinson’s symptoms are severe
  + *Parkinson’s patients are at increased risk for falls – orthostatic hypotension is a common adverse effect*
* Assess ability to carry out ADLs at home and explore the need for referrals
* Evaluate home safety needs
* Teach to rise from lying to sitting or standing slowly to avoid dizziness/falls
  + *Hypotension is a side effect of Sinimet*
* Teach to watch for and report immediately any signs of changes in mood
  + i.e. – increased aggression/confusion
  + *These drugs can cause mood disturbances and increase suicidality*
* **Instruct to take med on an empty stomach or to avoid taking with a high-protein meal**
  + ***May impair absorption***
* **Teach to avoid excessive consumption of B6 (bananas, wheat germ, fortified cereals, green vegetables, meat, legumes, and of course multivitamins containing it)**
  + ***May impair absorption***
* Teach about importance of returning for follow-up lab studies
  + *If* ***hepatic and renal function*** *decrease, this may slow the metabolism and excretion of the drug, which can lead to OD or toxicity*
* Advise that urine or sweat may darken, so to use protective measures to avoid staining clothes

Prototype drug Benztropine (Cogentin) Adverse Effect /Interactions------------------------------ 1

* Causes insomnia – “so bring your sleeping pills” x\_x
* Adverse Effects
  + Typical anticholinergic side effects such as dry mouth, constipation, and tachycardia.
  + Sedation, drowsiness, dizziness, restlessness, irritability, nervousness, **insomnia**
  + Contraindicated in narrow-angle glaucoma, myasthenia gravis, and obstructive GI/GU diseases
* Interactions
  + Drug-Drug
    - Alcohol, TCAs, MAOIs, phenothiazines, procainamide, quinidine
      * Combined sedative effects
    - OTC cold medicines
    - Other drugs that enhance dopamine release or activate dopamine receptor
    - Haloperidol (decreases effectiveness)
    - Antihistamines, phenothiazines, TCAs, disopyramide phosphate, wuinidine
      * May increase anticholinergic effects
    - Antidiarrheals
      * May decrease absorption

**Chapter 21- Drugs for Neuromuscular disorder**

Prototype drug: Dantrolene mechanism of action/Adverse effects-----------------------------------1

* Actions & Uses
  + Spasticity, especially spasms of the head/neck
  + It directly relaxes spasms by **interfering with the release of calcium ions from storage areas in skeletal muscle cells (NOT cardiac or smooth muscle)**
  + Especially useful for spasms r/t spinal injury, stroke, cerebral palsy, MS, occasionally muscle pain after heavy exercise, and malignant hyperthermia
* **Adverse Effects**
  + Muscle weakness, drowsiness, dry mouth, dizziness, nausea, diarrhea, tachycardia, erratic blood pressure, photosensitivity, urinary retention, fatigue

Table 21.1 Baclofen, clonazepam, Flexeril-Adverse effects 1

* Non-underlined: *Drowsiness, dizziness, urinary retention*
* Underlined: Respiratory depression and CV collapse
  + Edema of tongue, anaphylactic reaction, coma, laryngospasm

Table 21.2 Skeletal muscle Direct acting Antispasmodic Adverse effect/labs 1

* Dantrolene
  + Adverse effect = hepatic necrosis
  + Labs = LFTs (AST/ALT)
* Botox
  + Adverse effect = anaphylaxis, dysphagia, death
  + Labs = Should not affect labs (effects should be local)

Prototype Dantrolene Contraindications/Interactions 2

* Contraindications
  + Patients with impaired cardiac or pulmonary function or hepatic diseases
* Interactions
  + Calcium channel blockers (i.e. Verapamil) 🡪 V-fib and CV collapse
  + OTC cough medications and antihistamines, alcohol, other CNS depressants

Prototype Flexeril Action/ Adverse effect/overdose Tx 1

* **Action**
  + Depresses motor activity primarily in the brainstem
  + Limited effects in the spinal cord
  + Increases circulating levels of NE, blocking presynaptic uptake
* **Adverse effects**: look at Table 21.1 (underlined)
  + Edema of tongue, anaphylactic reaction, respiratory depression, coma, laryngospasm, CV collapse
* **OD Tx**
  + physostigmine

Skeletal muscle relaxants mechanism of action 1

* Inhibit upper motor neuron activity within CNS, causing CNS depressant effects or altering simple spinal reflexes

~~Mechanism of action of centrally acting agent and direct acting antispasmodics 1~~

* Question removed

Treating muscle spasms directly at muscle tissue- Botulinum toxin 1

* In large doses, it acts as a poison – it is the bacteria responsible for food poisoning
* At lower doses it is safe and effective; used to treat dystonia
* Mechanism of action: blocks the release of ACh from cholinergic nerve terminals (the NT responsible for voluntary muscle contraction)
* Injections are given with local anesthetic to reduce pain
* Therapy is associated with extreme muscle weakness, so it is often only applied to small muscle groups, or with centrally-acting oral meds to increase function of a range of muscle groups

**Chapter 49**

Mechanism of action of cycloplegic drugs 1

* Cycloplegic drugs dilate the pupil and paralyze the ciliary muscle and prevent the lens from moving
* Occupies muscarinic cholinergic receptors, blocking the PSNS actions of ACh (thereby inducing fight-or-flight symptoms)
* They increase IOP, so their use is contraindicated in glaucoma patients

Pharmacotherapy for minor eye conditions 1

* These are drugs for minor irritation and dryness; some lubricate only the surface of the eye, whereas others penetrate and affect a specific area.
* Irritation
  + Vasoconstrictors often used (one example is Visine)
* Conjunctivitis
  + Topical corticosteroids and NSAIDs
  + For allergic conjunctivitis, antihistamines and mast cell stabilizers are the preferred treatment; they do not cause excessive drying of the eye.

Prototype Latanoprost Administration alert/Adverse effect 2

* Class
  + Prostaglandin analog
* **Administration Alert**
  + Remove contact lenses before instilling, and don’t put them back in for 15 minutes
  + Avoid touching the eye or lashes with any part of the dropper to avoid cross-contamination
  + Wait 5 minutes before/after instillation of a different eye drop
* **Adverse Effects**
  + Ocular symptoms, such as conjunctival edema, tearing, dryness, burning, pain, irritation, itching, sensation of foreign body in eye, photophobia, visual disturbances
  + Eyelashes may get thicker/darker
  + Heightened pigmentation may occur (blue iris turns brown)

Correct method of administration of eye drops 1

* Instilled into the conjunctival sac
* Lacrimal duct area held with gentle pressure for 1 minute to prevent systemic effects
* Waiting 5 minutes between multiple medications

Medication for middle ear infection 1

* Middle ear infection (Otitis media) is commonly associated with URIs, allergies, or auditory tube irritation.
* It is treated with a course of systemic rather than topical antibiotics (Amoxicillin is prescribed for most children)
* It may also warrant treatment for pain, edema, and itching. These are treated with topical corticosteroids mixed with antibiotics (like ciprofloxacin and dexamethasone (CiproDex) or neopoly HC (Cortisporin)). Acetaminophen/NSAIDs may also be used for fever/pain.

Prototype drug timolol: Interactions 1

* Class
  + Miotic; beta adrenergic antagonist
* Interactions
  + May result if significant systemic absorption occurs
  + Use timolol cautiously in those on other **beta-blockers** due to additive cardiac effects
  + **Bradycardia and hypotension** may result when it is used with anticholinergics, **nitrates, reserpine, methyldopa, or verapamil**
  + Hypertension followed by severe bradycardia may result from concurrent use of epinephrine

Table 49.3 Otic Preparation Carbamide peroxide, Polumyxin ALL Adverse effects 2

* Allergic reactions (antibiotics), *ear irritation, local stinging or burning, dizziness*

**Chapter 14 Anxiety & Insomnia**

~~Area responsible for sleep and wakefulness ------------------------------------1~~

* Question removed

~~Indication for the need of pharmacotherapy in patient with anxiety------2~~

* Question removed

~~Treating anxiety and insomnia with CNS agent----------------------------------1~~

* Question removed

Table 14.3 SSRIs------Fluoxetine---Side effect/Adverse effect----------------1

* Underlined ones
  + Stevens-Johnson Syndrome, extreme mania/hypomania and suicidality, abnormal bleeding, extreme psychomotor disturbances, seizures, ANS instability with rapid fluctuations of VS, severe hyperthermia, serotonin syndrome
* *Nausea, vomiting, dry mouth, insomnia, somnolence, headache, nervousness, anxiety, GI disturbances, anorexia, sexual dysfunction, agitation, dizziness, fatigue*

Antidepressants-------MAOIs ---------------------------------------------------------1

* Don’t eat tyramine-containing foods
* Increased NE 🡪 HT crisis

Prototype Drug Escitalopram Action and Uses/Interactions-----------------1

* Action and Uses
  + **SSRI** 
    - No NE action, therefore no sympathomimetics/anticholinergic activity
  + Labeled for GAD and depression; unlabeled use for panic disorders
* Interactions
  + DON’T GIVE anywhere near an **MAOI!**
    - This can cause serotonin syndrome (ANS hyperactivity, hyperthermia, rigidity, diaphoresis, and NMS)
    - It can also cause hypertensive crisis
  + It increases plasma levels of metoprolol and cimetidine
  + Don’t use with alcohol/other CNS depressants due to increased effects
  + **St. John’s Wort** may cause SS\

Table 14.4 ~~Clonazepam~~ Diazepam------ Adverse effect--------------------------------------1

* Acute hyperexcited states, hallucinations, increased muscle spasticity, renal impairment, congenital defects among women who are pregnant, respiratory impairment due to hypersalivation, respiratory depression, laryngospasm, CV collapse

Table 14.4 Lorazepam------ Adverse effect----withdrawal----------------------1

* **In general – the return of anxiety and insomnia**
* Withdrawal: fever, psychosis, seizures, increased HR, lowered BP, loss of appetite, muscle cramps, impairment of memory, concentration, and orientation, abnormal sounds in the ears, blurred vision, insomnia, agitation, anxiety, panic
* OD? – give flumazenil (Romazicon)

Table 14.5 Antiseizure medication-----------Adverse effect-------------------1

* Phenobarbital
* Agranulocytosis, respiratory depression, SJS, exfoliative dermatitis (rare), CNS depression, coma, death, ….Laryngospasm

Prototype drug Zolpidem----------Adverse effects/Interaction-Herbal & Food—1

* Adverse Effects
  + Daytime sedation, confusion, amnesia, dizziness, depression, nausea, vomiting
* Herbal & Food Interaction
  + If it is taken with food, the absorption slows
  + “eat after 8, and your Ambien won’t work so great”

Nursing implication----------------------------------------------------------------------1

* Implementation, Minimizing adverse effects
  + Assess for changes in visual acuity, blurred vision, loss of peripheral vision, seeing rainbow halos around lights, acute eye pain, or any of these symptoms accompanied by nausea and vomiting
    - Increased intraoptic pressure in patients with narrow-angle glaucoma may occur in patients taking benzodiazepines
  + Monitor affect and emotional status
    - Drugs may increase risk of mental depression, esp in patients with suicidal tendencies
    - Concurrent use of alcohol and other CNS depressants increase the effects and the risk
  + Encourage appropriate lifestyle changes – lowered caffeine intake including OTC meds that contain it, increased exercise during the day but not immediately before bedtime, limited or no alcohol intake, smoking cessation
    - Healthy lifestyle changes will support and minimize the need for drug therapy.
    - Caffeine and nicotine may decrease the effectiveness of the drug
    - Alcohol and other CNS depressants may increase the adverse effects of the drugs
  + Avoid abrupt discontinuation of therapy
    - Withdrawal symptoms, including rebound anxiety and sleeplessness, are possible with abrupt discontinuation after long-term use

**Chapter 19: Anesthesia**

Stages of general anesthesia----------------------------------------------------------------------1

* Stage 3 is the surgical state – most important for us to know

|  |  |
| --- | --- |
| **Stage** | **Characteristics** |
| Stage 1 | * loss of pain; * patient loses gen. sensation but may be awake * this stage proceeds until the patient loses consciousness |
| Stage 2 | * Excitement & Hyperactivity * Patient may be delirious & try to resist treatment * HR & breathing may become irregular * BP can increase * IV agents are administered here to calm the patient |
| Stage 3 | * Surgical anesthesia * Skeletal muscles become relaxed and delirium stabilizes * Cardiovascular & breathing activities stabilize * Eye movements slow * Patient becomes still * Surgery begins & remains until the procedure ends |
| Stage 4 | * Paralysis of the medulla region of the brain (responsible for controlling respiratory & CV activity) * If breathing or the heart stops, death could result * This stage is usually avoided during gen. anesthesia |

Topical Anesthetics----------------------------------------------------------------------------------1

* Creams, sprays, suppositories, drops, and lozenges
* Applied to mucous membranes including the eyes, lips, gums, nasal membranes, and throat
* Very safe unless absorbed
* Apply only over a small area of the skin to avoid systemic absorption

Table 19.4 Inhaled anesthetics Gas adverse effect------------------------------------------1

* Nitrous oxide – malignant hyperthermia, apnea, cyanosis

Prototype drug Succinylcholine----------------action and uses/Adverse effect----------1

* Mechanism of Action
  + It acts on cholinergic receptor sites at NMJ (just like ACh)
  + At first, depolarization occurs (skeletal muscles contract)
  + After repeated such contractions, the membrane is unable to repolarize as long as the succinylcholine remains attached to the receptor
  + Effects:
    - first noted as muscle weakness and muscle spasms
    - followed by paralysis
  + When the IV infusion of succinylcholine is stopped, the effects wear off within a few minutes – this is because the drug is rapidly broken down by cholinesterase
  + Why is it used? It stops muscle movement during procedures, and it reduces the amount of general anesthetic needed for procedures
  + Because it can cause malignant hyperthermia, dantrolene sodium (Dantrium) is used with it perioperatively to reduce the signs in susceptible patients
* Adverse Effects
  + Can cause complete paralysis of diaphragm and intercostal muscles (which necessitates the use of mechanical ventilation during surgery)
  + Bradycardia
  + Respiratory depression
  + Malignant hyperthermia
  + High doses 🡪 tachycardia, hypotension, urinary retention

Inhaled anesthetics---------------nursing implication------------------------------------------ 1

* …Report if BP below 90/60
* Know that there are different stages of anesthesia and what is expected from you when a patient is in each of these stages
* What should you do if the patient moves into stage 2? – Keep them calm and let them relax so that they can move into stage 3 as quickly as possible
* Inhaled anesthetics are excreted via the lungs – the nurse should encourage the patient to take deep breaths and move the lower extremities frequently in the post-op period to assist in removing the remaining anesthetic.
* Malignant hyperthermia is a rare but potentially fatal adverse effect of all inhalation anesthetics
* Nitrous oxide should be used cautiously in:
  + Patients with myasthenia gravis (because it may cause respiratory depression and prolonged hypnotic effects)
  + Patients with CVD, especially those with increased ICP (because the hypnotic effects of the drug may be prolonged or potentiated)
* Volatile Liquids
  + Some enhance the sensitivity of the heart to drugs such as Epi, NE, dopamine, and serotonin
    - Assess VS and potential for drug/drug interactions
  + Most depress CV and respiratory function

Spinal anesthesia---------post operative care-----priority nursing interventions---------1

* Top concern is vitals, because respiratory depression is a big concern, as is change in HR
* Malignant hyperthermia may occur as well but the PRIORITY is vitals
* Assess the ability to move limbs distal to the regional anesthetic
  + The patient may regain some motor ability before sensation returns
* Immediately report BP below 90/60, tachy/brady, changes in LOC, dyspnea, decrease in respiratory rate
  + Regional blocks may cause hypotension with reflex tachycardia
  + Bradycardia, hypotension, decreased LOC, decreased respiratory rate, and dyspnea may indicate that the anesthetic has entered systemic circulation and is acting as a general!
* Note that a patient is at risk for falls post-anesthesia – so they should call for help prior to getting out of bed or walking alone
* Monitor for the onset of pain as the block wears off
* Note, spinal anesthesia can cause a spinal headache

Table 19.1 Methods of local anesthetics administration-------------------------------------1

* How is an epidural, nerve block, spinal anesthetic given?

|  |  |  |
| --- | --- | --- |
| **Route** | **Formulation/Method** | **Description** |
| **Epidural anesthesia** | Injection into the epidural space of the spinal cord | Most commonly used in obstetrics during labor & delivery |
| Infiltration (field block) anesthesia | Direct injection into tissue immediate to the surgical site | Drug diffuses into tissue to block a specific group of nerves in a small area close to the surgical site |
| **Nerve block** Anesthesia | Direct injection into tissue that may be distant from the operation site | Drug affects nerve bundles serving the surgical area; used to block sensation in a limb or large area of the face |
| **Spinal Anesthesia** | Injection into the cerebral spinal fluid (CSF) | Drug affects a large, regional area such as the lower abdomen & legs You would give this during a C-section (not a vaginal birth) |
| Topical (surface) anesthesia | Creams, sprays, suppositories, drops, & lozenges | Applied to mucous membranes including the eyes, lips, gums, nasal membranes, and throat; very safe unless absorbed |

Table 19.4 halothane---------------------------adverse effect------------------------------------1

* Myocardial depression, marked hypotension, pulmonary vasoconstriction, hepatotoxicity

Table 19.5 Propofol/midazolam/~~ketamine~~-----adverse effect-------------------------------1

* Propofol (Diprivan) (barbiturate-like agent)
  + Circulatory or respiratory depression with apnea, laryngospasm, anaphylaxis
* Midazolam HCl (Versed) (benzodiazepine)
  + Cardiovascular collapse, laryngospasm

**Chapter 34:**

Selection of Effective Antibiotic-----------------------------------------------------------------1

* To select one, you need to know C&S results
* Sometimes, therapy will begin with a broad-spectrum antibiotic, and then possibly changed to a narrow-spectrum antibiotic after C&S results are in
  + Antibiotics should generally not be combined, as antagonism may occur, and may promote resistance
  + Multiple-drug therapy may be warranted in some situations (i.e. TB, HIV)

Table 34.2: Penicillin G sodium/potassium, Amoxicillin-Clavulanate------------------1

* Anaphylaxis symptoms, including angioedema, circulatory collapse and cardiac arrest; nephrotoxicity

Prototype Drug Penicillin G sodium/potassium Interactions----------------------------1

* Drug-Drug
  + Decreases the effectiveness of oral contraceptives
  + Colestipol will decrease the absorption of penicillin
  + Potassium-sparing diuretics may cause hyperkalemia
* Lab tests
  + May cause positive Coombs test and false positive urine or serum proteins

Table 34.3: Cephalosporins Cephalexin, cefepime Adverse effects--------------------1

* **Cefotaxime**
  + *Diarrhea, abdominal cramping, nausea, fatigue, rash, pruritus, pain at injection sites, oral or vaginal candidiasis*
  + Pseudomembranous colitis, nephrotoxicity, anaphylaxis

Prototype: Tetracycline administration alert/Interactions-------------------------------1

* **Administration Alerts** 
  + Administer oral drug with full galss of water to decrease esophageal and GI irritation
  + Administer tetracycline and antacids 1-3h apart
  + Administer antilipidemic agents at least 2 hours before or after tetracycline
  + Pregnancy Category D
* **Interactions**
  + Drug-Drug
    - Milk products, magnesium-containing laxatives, antacids, colestipol, cholestyramine
      * decrease absorption
    - Oral contraceptives
      * Decreases their effectiveness
  + Lab Tests
    - May increase BUN, AST, ALT, amylase, bilirubin, and alkphos
  + Herbal/Food
    - Dairy products

Prototype: Erythromycin Action and Uses/~~Adverse effects~~------------------------------1

* It is inactivated by stomach acid and is thus formulated as coated, acid-resistant tablets or capsules that dissolve in the small intestine
* Its main application is for patients who are unable to tolerate penicillins or who may have a penicillin-resistant infection
* It has a spectrum similar to that of the penicillins and is effective against most gram-positive bacteria
* It is often a preferred drug for infections by *Bordetella pertussis* (whooping cough), and *Corynebacterium diphtheriae*

Prototype: Gentamicin Adverse effect/Administration alert-----------------------------1

* **Administration Alerts**
  + For IM administration, give into a large muscle
  + Use only IM and IV drug solutions that are clear, colorless, or slightly yellow. Discard discolored solutions or those that contain particulate matter
  + Withhold the drug if the peak serum level lies above the normal range of 5-10mcg/mL
  + Pregnancy Category C
* **Adverse Effects**
  + Most frequent: rash, nausea, vomiting, fatigue
  + Ototoxicity (early signs: tinnitus, vertigo, persistent headaches) – may produce loss of hearing or balance that may become permanent with continued use
  + Nephrotoxicity (oliguria, proteinurea, elevated BUN & Cr)
  + Resistance – and cross-resistance between other aminoglycosides

Prototype: Ciprofloxacin: Adverse effects/~~Contraindications~~ ----------------------------1

* Most frequent: nausea, vomiting, diarrhea
  + Administration with food may diminish these effects
  + …but NOT antacids or mineral supplements, since absorption will be diminished
* Less common: phototoxicity, headache, dizziness
* Black Box Warning: **Tendon inflammation and rupture**
  + Any complaints of difficulty walking or pain in the foot or leg should be immediately reported

**Chapter 35:**

Mechanism of action of antifungal drugs-------------------------------------------------------1

* Bacteria and fungi have different physiologies – in fact, fungal cells are more similar to human cells than they are to bacteria!
* One main difference between human cells and fungal cells is that human cells have cholesterol in their cell membranes, whereas fungal cells have ergosterol
* Many antifungals work by inhibiting the synthesis of ergosterol, which **causes the plasma membrane to become porous and leaky**
  + Amphotericin B, terbinagine, and nystatin work in this way
* Other antifungals use enzymatic pathways instead – targeting enzymes that fungal cells have but human cells do not and converting them into toxins

Prototype: Amphotericin B: Administration alert/Interaction------------------------------1

* Administration Alert
  + **Infuse slowly,** because CV collapse may otherwise result
  + Administer premedication to decrease the risk of infusion reactions
  + If the BUN is > 40mg/dL or creatinine is > 3mg/dL, HOLD THE FUNGIZONE!
  + Pregnancy Category B
* Interaction
  + Drug-Drug
    - Many!
    - Drugs that reduce renal function (aminoglycosides, vancomycin, coarboplatin)
    - Corticosteroids, skeletal muscle relaxants, thiazole (these may potentiate hypokalemia
    - Digoxin (may increase the risk of digoxin toxicity in those with pre-existing hypokalemia)
  + Lab Tests
    - May increase BUN, creatinine, alk phos, AST, ALT
    - May decrease K, Ca, and Mg
  + (Herbal/Food unknown)

Nursing Process: Antifungal drugs-Implementation-------------------------------------------1

* Continue to monitor for signs of ototoxicity
  + Antifungals may cause ototoxicity and require frequent monitoring to prevent adverse effects
* Continue to monitor for signs of hepatic toxicity (jaundice, RUQ pain, darkened urine, diminished urine output, tinnitus, vertigo) in patients on IV or oral antifungals
  + Antifungals may cause hepatic toxicity and require frequent monitoring to prevent adverse effects
* Monitor the IV site frequently for any signs of extravasation or thrombophlebitis
  + IV antifungals are irritating to veins. Use a central line if possible or frequently monitor the IV site.
  + Infusion pumps must be used to ensure the proper dosage rate and prevent excessive flow rate.

Prototype: Fluconazole: Adverse effects----------------------------------------------------------1

* Nausea, vomiting, diarrhea at high doses
* **Hepatotoxicity** (rare)
* **SJS** (in patients with immunosuppression)

Table 35.4 Griseofulvin, Nystatin Adverse effects--------------------------------------------1

* Griseofulvin
  + Granulocytopenia
* Nystatin
  + No specific effects noted….
* What it says in the book: “Granulocytopenia (griseofulvin), cholestatic hepatitis (oral terbinafine), neutropenia (oral terbinafine”

Table 35.5: ~~Chloroquine,~~ Quinine: Adverse effects--------------------------------------------1

* Conchonism (tinnitus, ototoxicity, vertigo, fever, visual impairment), hypothermia, coma, CV collapse, agranulocytosis

Nursing Process: Protozoan/Helminthic Infection: Assessment/Implementation------1

* Continue to monitor periodic lab work: hepatic and renal function tests, CBC, ECG, C&S, and fecal O&P.
  + Hepatic and renal labs, particularly with IV therapy, should be monitored to prevent adverse effects
  + Periodic C&S tests or fecal O&P tests may be ordered if infections are severe or are slow to resolve to confirm appropriate therapy
  + ECG monitoring may be required with some antimalarials
* Monitor for S/S of neurologic effects (dizziness, drowsiness, and headache) and ensure patient safety. Be cautious with the elderly who may be at increased risk for dizziness and falls
  + Teach the patient to rise from lying or sitting to standing gradually if dizziness occurs

Prototype Drug: Mebendazole Interaction-----------------------------------------------------------------1

* Drug-Drug
  + Carbamazepine and phenytoin (they cause increased metabolism of mebendazole)
* Herbal/Food
  + High-fat foods may increase the absorption of the drug

**Chapter 37:**

Growth Fraction-----------------------------------------------------------------------------------------1

* Ratio of replicating cells to resting cells
* Some normal tissues have high growth fractions, so are more susceptible to the toxic effects of chemotherapy
* Cancers with a low growth fraction (less sensitive to antineoplastics)
  + Solid tumors (breast, lung)
* Cancers with a high growth fraction (more sensitive to antineoplastics)
  + Certain leukemias and lymphomas
* Normal tissues with a high growth fraction
  + Hair follicles
  + Bone marrow
  + GI epithelium

Special Pharmacotherapy Protocols and strategies for cancer chemotherapy----------1

* The problem:
  + Tumor cells have a high mutation rate; as such, tumors become increasingly heterogenous with continued growth
  + This means that antineoplastics might not affect some tumor cells at all – because the cells are different from each other, and can also develop resistance to antineoplastics
    - In other words, tumors may become refractory (resistant) to treatment
* The solution:
  + Combination chemotherapy – using drugs from different antineoplastic classes
    - Different drugs affect different stages of the cancer cells’ cycle
    - Different drugs have different mechanisms of action
    - This increases the percentage of cell kill
    - Using multiple drugs allows for lower doses of each
      * this reduces toxicity and slows the development of resistance
  + Specific dosing schedules
    - Optimal timing between doses to allow for maximum effectiveness
      * Waiting allows tumor cells that were not dividing during the previous treatment to begin doing so – making them susceptible to the therapy
      * Recovery of healthy cells
        + Including recovery from adverse effects such as bone marrow suppression

Table 37.4 Alkylating Agent Cyclophosphamide:Adverse effect-----------------------------1

* Bone marrow suppression (neutropenia, anemia, thrombocytopenia), severe nausea and vomiting, diarrhea, SJS, hemorrhagic cystitis, pulmonary toxicity, hypersensitivity reactions (including anaphylaxis), nephrotoxicity
* Other underlined effects: neurotoxicity (carboplatin, cisplatin, oxaliplatin), ototoxicity (cisplatin)

Prototype Cyclophosphamide Administration Alert------------------------------------------------1

* Dilute prior to IV administration
* **Monitor platelets prior to IM administration – if low, hold the dose**
* To avoid GI upset, take with meals or divide doses
* Pregnancy Category C

Table 37.5 Methotrexate Adverse effects----------------------------------------------------------1

* Bone marrow suppression (neutropenia, anemia, thrombocytopenia), severe nausea, vomiting, and diarrhea, hepatotoxicity, mucositis, pulmonary toxicity, hypersensitivity reactions (including anaphylaxis)
* Other underlined effect: neurotoxicity (cytarabine, fluoracil, fludarabine, cladribine)

Prototype Methotrexate Action and Use/Administration alert-------------------------------1

* Action and Use
  + Blocks the synthesis of folic acid (vitamin B9)
    - It therefore inhibits replication, particularly in rapidly dividing cells
  + Rx: for choriocarcinoma, osteogenic sarcoma, leukemias, head and neck cancers, breast carcinoma, lung carcinoma
    - It is also used as an immunosuppressant for RA, ulcerative colitis, lupus, and psoriasis that are unresponsive to safer medications
* Administration Alert
  + Avoid skin exposure to drug
  + Avoid inhaling drug particles
  + Dilute prior to IV administration
  + Pregnancy Category X

Table 37.7 vincristine Adverse effect----------------------------------------------------------------1

* Bone marrow suppression (neutropenia, anemia, thrombocytopenia), severe nausea and vomiting, diarrhea, cardiotoxicity, mucositis, pulmonary toxicity, hypersensitivity reactions (including anaphylaxis), neurotoxicity, nephrotoxicity

Table 37.8 Hormones Dexamethasone-------------------------------------------------------------1

* Thrombophlebitis, muscle wasting, osteoporosis
* Other underlined effect: hepatotoxicity (testosterone, testolactone)